tion rather than obliteration of the pneumothorax.

It is believed that total decortication of the pneumothorax pocket was advantageous from the technical as well as the therapeutic point of view.

In the first case it was debated whether to perform thoracoplasty with the intent of obliterating the space. In view of the operative finding of thick peel over the chest wall and upper mediastinum it is doubtful that it would have been successful.

In the second case a combination of closed intercostal drainage, phrenic nerve interruption and pneumoperitoneum was contemplated but considered contraindicated in view of the obvious pulmonary suppuration in the lower and middle lobes, because the chance of thus effecting relief of the attacks of fever, cough and expectoration seemed remote. Likewise the threat of empyema developing seemed great.

SUMMARY

The case histories of two patients with limited or localized chronic tension pneumothorax are presented. The cause of the pneumothorax in one case was probably suppurative pneumonitis with localized effusion and in the other probably rupture of an emphysematous bleb. In both patients total lobar collapse and suppuration were present and both were treated by complete decortication and resection of the involved lobes.

1205 Medico-Dental Building, 233 A Street, San Diego 1.

REFERENCES

1. Brewer, III, L. A., Dolley, F. S., and Evans, B. H.: The surgical management of chronic "spontaneous" pneumothorax, J. Thor. Surg., 19:167, 1950.

2. Briggs, J. N., Walter, R. W., and Byron, F. X.: Spontaneous pneumothorax, Dis. of Chest, 24:564, 1953.

3. Brock, R. C.: Recurrent and chronic spontaneous pneumothorax, Thorax, 3:88, June 1948.

4. Goldschmidt, W.: Phrenicusausschaltung beim pathologischen Pneumothorax, Wein. klin. Wchnschr., 44:1276, 1931.

5. Hennell, H., and Steinberg, M. F.: Tense pneumothorax: Treatment of chronic and recurrent forms by induction of chemical pleuritis, Arch. Int. Med., 63:648, 1939.

6. Horsley, J. S., and Bigger, I. A.: Lung Decortication and Pneumothorax, p. 685, Operative Surgery, C. V. Mosby Co., 5th ed., 1940.

7. Hoyer, L. P., and Claggett, O. T.: Treatment of chronic spontaneous pneumothorax by lobectomy, J. Thor. Surg., 15:418, 1946.

8. Kipfer, R.: Kasuistischer Beitrag zur Pathogenese des Spontanpneumothorax und zu seiner Therapie mit Hilfe der Thorakoskopie und endopleuralen Kaustik, Verhandl. d. deutsch. Gesellsch. f. inn. Med. Kong., 44:193, 1932.

9. Medici, F. A.: Resultados de la pleurodesis por el metodo de Bethune en el neumotorax espontaneo benigno, An. Cated. de pat. y clin. tuberc., 6:106, 1944.

10. Movitt, E. R., Smith, J. V., and Eloesser, L.: Treatment of spontaneous pneumothorax, Dis. of Chest, 13:221, 1947.

11. Sarot, I. A.: Extrapleural pneumonectomy and pleurectomy in pulmonary tuberculosis, Thorax, 4:173, 1949.

12. Tyson, M. D., and Crandall, W. B.: The surgical treatment of recurrent ideopathic spontaneous pneumothorax, J. Thor. Surg., 10:566, 1941.

Cortisone in Treatment of Trichinosis

JOSEPH F. SADUSK, JR., M.D., Oakland

WHILE TRICHINOSIS is generally regarded as a benign disease, it is well known that the clinical course may at times be quite severe; and indeed mortality rates of from approximately 3 to 6 per cent have been recorded.[3, 6, 9]

Until very recently, the only recognized therapeutic procedure was that of bed rest and supportive medication. Scattered reports on the use of corticotropin (ACTH)[4] and cortisone[7, 8] indicate that these agents may be of considerable value in the treatment of trichinosis.

The purpose of this communication is to report the treatment of a case of trichinosis with cortisone, to show that this agent was most effective in the amelioration of symptoms, and to attempt to define the appropriate dosage of the drug.

REPORT OF A CASE

A 31-year-old traveling salesman was admitted to the hospital on the thirteenth day of illness with complaints of fever, chills, muscle aching and pain, sweats, severe frontal headache and swelling of the eyelids.

The illness began November 27, 1952, with a sudden severe chill and temperature rise to approximately 101° F. The patient then perspired profusely throughout the night. The next day he felt well and was essentially asymptomatic until December 2, at which time he noticed puffiness of the lower eyelids which rapidly spread in a day or so to the upper lids and finally involved the entire periorbital area. On December 5, the patient suddenly had another chill. The temperature rose rapidly, and there was muscle aching of a generalized nature but with particular severity in the anterior thigh muscles. On December 6 another severe shaking chill occurred and chills and aching of the muscles continued but the periorbital edema began subsiding slowly. Rather severe frontal headache developed. Temperatures ranged between 100.2° and 102.4° F. when he was admitted to hospital. There was no history of skin eruption or diarrhea.

The only infectious disease noted in the patient's history was measles. The patient, traveling by automobile throughout the rural districts of California, frequently ate inadequately cooked "hamburgers," possibly containing pork, at roadside stands.

Upon physical examination the patient was observed to be well developed and well nourished. He appeared acutely ill and prostrated. The temperature was 102.0°, the pulse rate 96, and the blood pressure 120/40 mm. of mercury. The skin was flushed, sweating and hot. No skin eruption was noted. There was a moderate tenderness of the anterior muscles of the thigh. The conjunctivae were clear. Moderate bilateral periorbital edema was

From the Medical Service, the Samuel H. Merritt Hospital, Oakland 9.

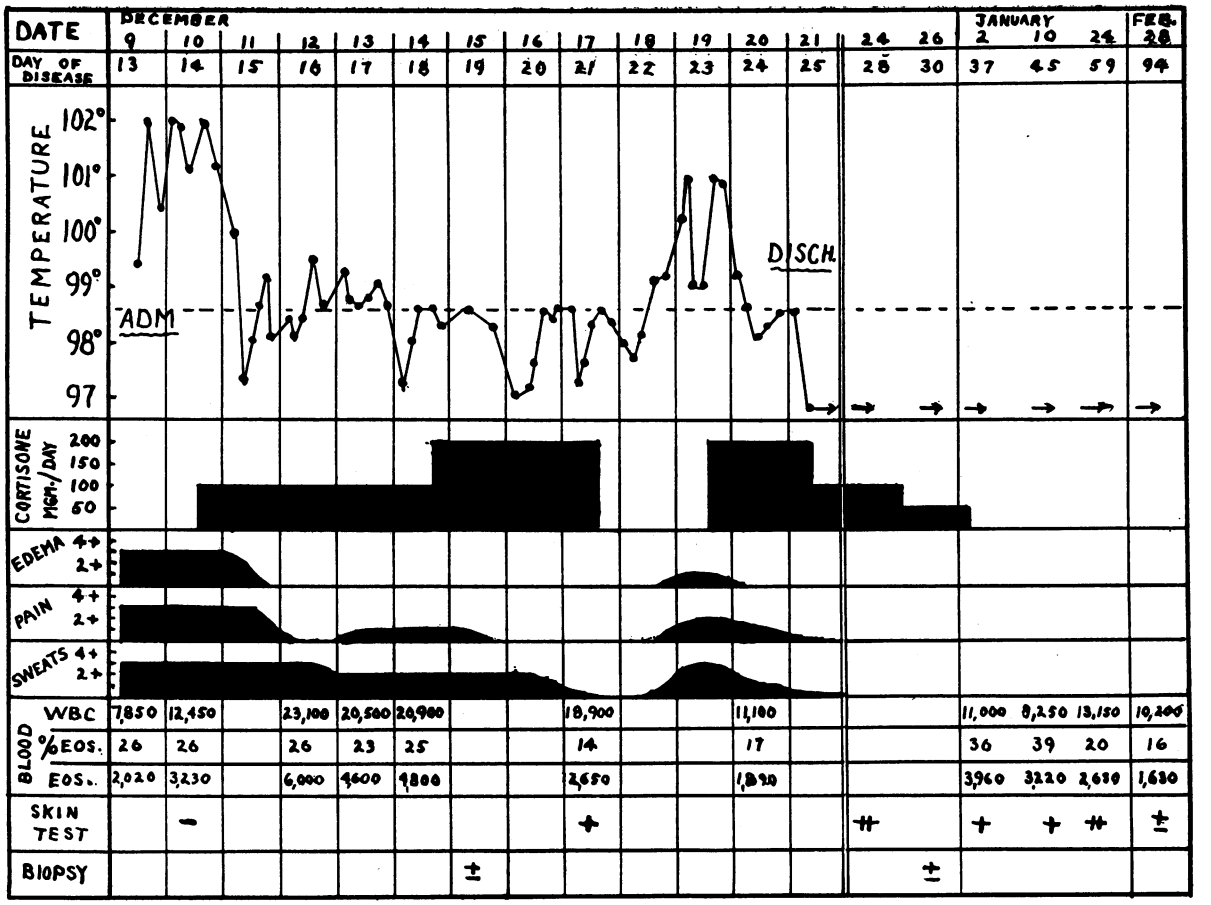

Chart 1.—Effect of cortisone in case of trichinosis.

noted. Pulsation was regular and bilaterally equal. The heart was not enlarged and there were no murmurs. The lungs were clear. The abdomen was soft and nontender and neither liver nor spleen was palpable. Deep reflexes were normal and equal, and there were no clinical signs of meningeal irritation.

Data on the subsequent course are shown in Chart 1. On admission, the following laboratory data were obtained. Erythrocytes numbered 4,470,000 per cu. mm. of blood and the hemoglobin content was 15.4 gm. per 100 cc. Leukocytes numbered 7,850 per cu. mm.—4 per cent nonsegmented neutrophils, 45 per cent segmented neutrophils, 20 per cent lymphocytes, 5 per cent monocytes and 26 per cent eosinophils. The urine was clear, with acid reaction, and the specific gravity was 1.020. It was negative for sugar and albumin. Centrifuge sediment showed only occasional granular casts. A culture of blood was sterile. Brucella agglutination was negative in a dilution of 1:20. The Widal reaction was as follows: B. typhosus 1:40 (H antigen) and negative (0 antigen), B. paratyphosus A negative, and B. paratyphosus B 1:40.

The Weil-Felix reaction (Proteus OX19) was negative in a titer of 1:20. Skin tests with a 1:10,000 dilution of trichinella extract (Lederle) did not cause a wheal with either the control or the antigen.

After 24 hours during which fever and severe symptoms and signs of muscle pain, sweats and periorbital edema continued, cortisone therapy was started with a dosage of 25 mg. by mouth every 6 hours for a total of 100 mg. per day. Within 12 hours, the temperature began to fall rapidly and by the end of 24 hours the periorbital edema, prostration and muscle pain had disappeared, although night sweats continued in diminishing degree for another six days. When low grade fever began on the second day of therapy and continued for another 24 hours, cortisone was increased to 200 mg. per day in a dosage of 50 mg. every six hours. The temperature promptly dropped to normal limits and the patient continued quite asymptomatic.

On December 15, the nineteenth day of disease, specimens of tissue were removed* from the anterior thigh muscles for study. On routine examination of about 35 sections of the material† no encysted parasites were seen but there was a focal inflammatory reaction between the muscle fibers representing myositis and associated angiitis. There were no vascular changes suggestive of periarteritis. The entire block was sectioned and every tenth section was reviewed but still no Trichina were found.

* By Dr. Walter L. Byers of Oakland.

† By Drs. Charles Baker, Ruth Seale, and Bruno Gerstl of Oakland.

Finally, sections lying in approximation with sections of areas showing myositis were also studied and a fragment of a Trichina was discovered (Figure 1).

A skin test with trichinella antigen on the twenty-first day of disease revealed a faintly positive reaction with a blanched wheal 3 x 4 mm., without surrounding erythema, and the control was entirely negative. The eosinophil content of the blood slowly decreased as shown in Chart 1.

Cortisone was abruptly discontinued on December 17 (the twenty-first day of disease) in order to determine what therapeutic rôle this drug might have played. Within 24 hours, the temperature rose, reaching 101.0° F., and symptoms and signs of muscle pain, sweats and periorbital edema reappeared. Administration of cortisone was resumed in a dosage of 200 mg. per day. The response was again dramatic, with fall of temperature to normal and abatement of signs and symptoms. There was a further drop in the total eosinophil content to 1,890 cells per cu. mm.

The patient was finally discharged on December 21 (twenty-fifth day of disease) after 13 days of hospitalization. Convalescence was continued at home with a dosage of 100 mg. of cortisone per day (25 mg. every six hours). This dosage was continued until the twenty-ninth day of disease, following which a dosage of 50 mg. daily (25 mg. every 12 hours) was continued until the thirty-seventh day of disease.

A trichinella skin test using the same solution of antigen as before was repeated on the twenty-eighth day of disease and there was a moderately positive reaction, the wheal measuring 8 x 9 mm. with a small surrounding area of erythema. Biopsy from the deltoid muscle was repeated on the thirtieth day of disease, but again, while there was definite evidence of a myositis similar to that previously described for the first biopsy, no parasites were found in the muscle tissues on routine examination.

Data on the patient as an outpatient are shown in Chart 1. It will be seen that reaction to skin tests ranged between mild and moderate and that the eosinophil count rose to a value of 3,960 cells per cu. mm. when cortisone was discontinued, but subsequently fell so by the ninety-fourth day of disease the eosinophil count had fallen to 1,630.

It is perhaps pertinent to note that on the second hospital day an electrocardiogram revealed no abnormalities except for an unusually high degree of elevation of the ST segment in the Wilson unipolar precordial lead V-2 only. This was considered to be a borderline tracing. Electrocardiograms repeated seven days later and then after a two-week interval were normal, with no evidence of the ST segment elevation.

The patient was seen for the last time on July 6, 1953. The trichinella skin test reaction was quite negative. Leukocytes numbered 7,100 per cu. mm., with 6 per cent eosinophils, and the sedimentation rate was well within normal limits. The patient was in excellent health, as he had been for the preceding three months.

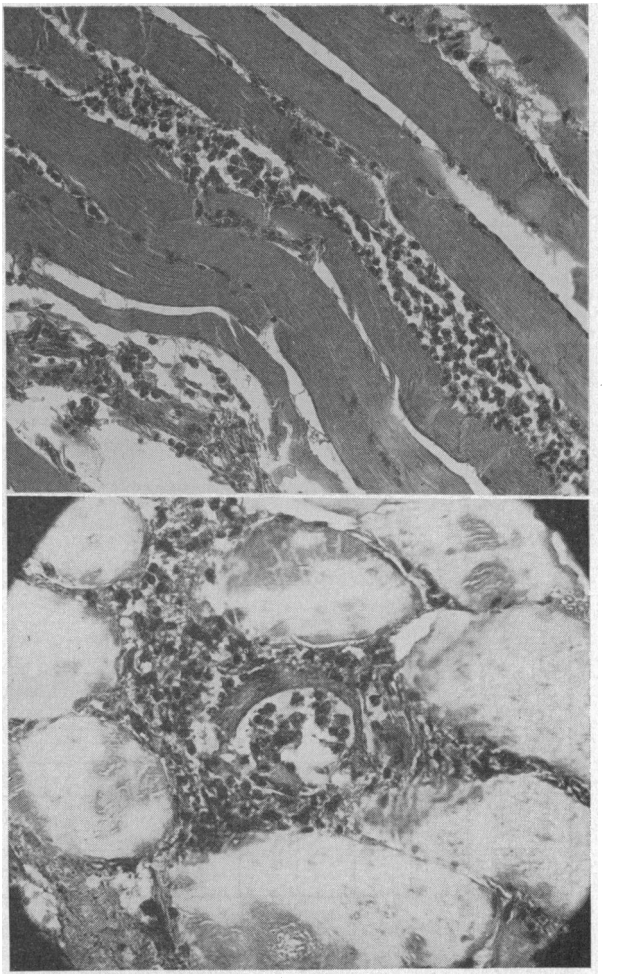

Figure 1.—Sections of anterior thigh muscle showing fragments of a trichina (upper) and associated myositis (lower).

COMMENT

There is no doubt that cortisone had a beneficial effect upon symptoms during the acute stage of the disease. The drop in temperature, relief from prostration, disappearance of periorbital edema and muscle pain, and development of a feeling of well-being were most dramatic and essentially in accord with the few previous reports in the literature. That these changes were due to cortisone is evidenced by the reappearance of symptoms when the medication was abruptly stopped on the twenty-first day of disease and eventual clearing again with resumption of medication.

It would appear that a dose of 100 mg. of cortisone per day was ineffective since the patient continued to have low grade fever, muscle pain and sweats when that amount was given. When the dosage was increased to 200 mg. per day, however, these symp-

toms rapidly cleared. Treatment should doubtless be prolonged, probably for a period of from two to three weeks, although this point is not yet sufficiently clarified. In the three case reports previously noted,[4, 7, 8] the use of corticotropin (ACTH) or cortisone was continued for from five to 18 days. It is likely that the duration of treatment depends upon the stage in which the disease is treated, with a longer period of therapy required for cases treated early in the disease.

The mechanism of action of corticotropin or cortisone in trichinosis—as well as in other diseases of bacterial or unknown origin—is not clear but probably depends upon an altered response between host and the infecting agent. In trichinosis, corticotropin and cortisone would appear to control the severity of disease until the larvae have become encysted and the host produces immune antibodies, thus rendering the disease clinically inactive.

It is clear from the studies of Luongo and coworkers[4] that corticotropin had no specific effect upon trichinella larvae in experimentally infected guinea pigs. On the other hand, those investigators found a definite reduction in the toxic effects of the disease in animals treated with corticotropin together with a temporarily diminished eosinophilia and a significantly longer survival period. Treated animals that died did so only after corticotropin was discontinued, which points up the necessity for a long term period of treatment.

Two additional points warrant further discussion, namely, the difficulty of securing confirmation of the disease by positive biopsy and the role that repeated injections of trichinella antigen may play in producing positive skin tests.

It is obvious that the intensive study leading to the discovery of a Trichina in the first biopsy material in the present study is not practical and consequently one must ordinarily be content with the finding of myositis. This point has already received mention in the literature.[1]

Whether or not repeated skin testing on several occasions with antigen will produce enough sensitivity to cause a positive skin reaction remains a controversial point. The experience of McCoy and co-workers[5] with repeated tests on control individuals indicates that the probability of sensitization is small; on the other hand, Baron and Brunner[2] showed that 56 per cent of test subjects had a positive reaction to skin test by the ninth test dose of Trichina antigen, and 33 per cent had sensitivity after three to six injections. Since it is unusual to persist in skin testing with trichinella antigen past two to three attempts, the importance of sensitization is rather minimal.

SUMMARY

Cortisone was efficacious in the treatment of a case of trichinosis insofar as the relief of signs and symptoms was concerned.

In the early and clinically active stage of the disease, apparently a dosage of 200 mg. daily of cortisone is required, with treatment continuing until the fourth or fifth week of disease in a diminishing dose down to 50 mg. per day.

459 Thirtieth Street, Oakland 9.

REFERENCES

1. Augustine, D. L.: Trichinosis—incidence and diagnostic tests, N.E.J.M., 216:463, March 18, 1937.

2. Baron, B., and Brunner, M.: Active sensitization in human beings with trichina antigen, J. Allergy, 13:459, July 1942.

3. Gould, S. E.: Trichinosis. Charles C. Thomas, Springfield, Ill., 1945, p. 279.

4. Luongo, M. A., Reid, D. H., and Weiss, W. W.: The effect of ACTH in trichinosis — a clinical and therapeutic study, N.E.J.M., 245:757, Nov. 15, 1951.

5. McCoy, O. R., Miller, J. J., Jr., and Friedlander, M.: J. Immunol., 24:1, Jan. 1933.

6. Ober, R. E.: Trichinosis—a review of cases in Massachusetts from 1936 to 1945, N.E.J.M., 235:839, Dec. 12, 1946.

7. Rosen, E.: Cortisone treatment of trichinosis, Am. J. Med. Sci., 223:16, Jan. 1952.

8. Rothenberg, F.: Treatment of trichinosis with cortisone, J. Med. Soc. N. J., 48:517, Nov. 1951.

9. Trichinosis (Editorial), J.A.M.A., 152:241, May 16, 1953.